

The NCVDLs REPORT



Veterinary News and Information From North Carolina's Diagnostic Laboratories

A Message from Our Director...

Fall is already upon us. It has been a wonderful summer, and although we welcome the cooler weather, we are left thinking how the season transpired so quickly! It has been a busy year at the diagnostic laboratory and we are making progress on many fronts. I will provide a brief update on a few topics/projects that are currently in the works.



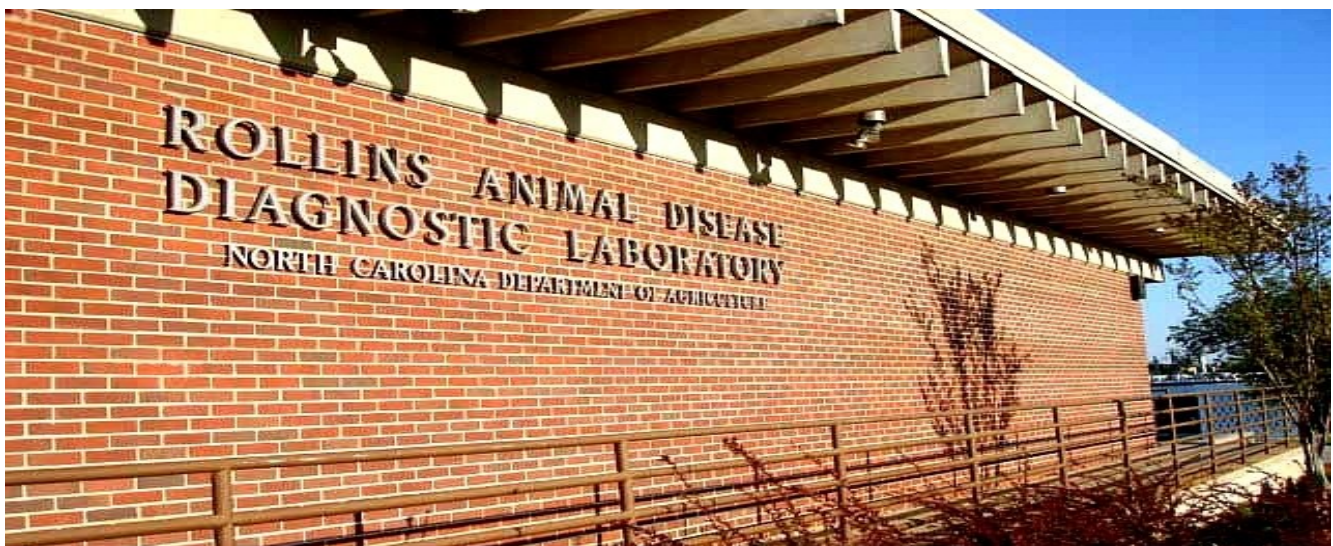
- ◆ We continue to make progress on the new home for the Rollins laboratory, the Agricultural Sciences Center! The Agricultural Sciences Center is a state-of-the art laboratory complex that will be built with \$94 million in funding from the Connect NC bonds approved by voters in March 2016.

The center will contain offices and labs that perform tests for four of the department's divisions: Veterinary, Food and Drug Protection, Standards, and Structural Pest Control and Pesticides. The building will replace four labs whose average age is more than 40 years. These labs are important to the state, and putting them in a central place will enable the department to be more effective and efficient. The three-story building will be built on the corner of Reedy Creek and Edwards Mill roads in Raleigh and has a projected completion date of late 2020.



- ◆ Thank you to everyone that took the time to participate in our user survey! We are currently evaluating the responses and comments and we will utilize the valuable information we received to ensure we are meeting your expectations and needs.
- ◆ We are excited to be currently advertising for the Bacteriology Section Head position. This position will oversee the bacteriologic testing at our facilities and provide interpretation on test results and cases. We have actively engaged with the NC State College of Veterinary Medicine and sought appropriate opportunities for collaboration to enhance our services by utilizing our combined resources. We are excited to announce that this position will be a dual-appointment between the diagnostic laboratory (75%) and the veterinary college (25%).
- ◆ We are currently evaluating our fee structure due to the rising costs of laboratory supplies. The NCVCLS is always examining ways to operate efficiently while continuing to support our valued customers and ensure they receive quality service and test results at a competitive price. Any fee changes that are instituted will ensure that we can maintain our capabilities to promote animal health and protect animal agriculture. It has been a number of years since our fees have been adjusted/restructured and we anticipate some changes to take effect in the coming months.
- ◆ Please be sure that your livestock and companion animals are up to date with veterinary care and vaccination status. This has been a busy year in North Carolina for Rabies cases in which confirmed cases in livestock are running higher than average. Also, mosquito born disease season is still with us and we have had several documented cases of Eastern Equine Encephalomyelitis within the last few weeks. These fatal diseases are preventable by appropriate vaccination!

Jim Trybus, DVM, DAVCP



To Necropsy or Not to Necropsy...

Unfortunately, a necropsy is an all too often underutilized procedure of veterinary medicine. I know the reasons behind this fact... it is hard to talk to an owner about a necropsy during an emotional euthanasia or sudden death of a patient, there is no time to talk to an owner about a necropsy during the hectic days we endure as practitioners, we're afraid to find out we were wrong about a diagnosis, or we have unanswered questions about what is involved and how to arrange a necropsy.

I practiced for 10 years before becoming a Veterinary Diagnostician at NCVDL. You know how many necropsies I performed while in practice? Three. Do you know how many deceased patients I sent for a necropsy? ZERO. Looking back, there are so many patients that I wish I had submitted for a necropsy. I know now that I would have gained valuable insight and knowledge that would have helped me with future cases.

A necropsy examination is an invaluable learning tool, that in a group made up of largely, analytical overachievers, we choose to ignore. When we can definitively determine a cause of death in a patient, we advance our skills and understanding to improve the field of veterinary medicine overall.

I know, sometimes, we just don't want to know. We don't want to know that we were wrong or feel like we guided a client down the wrong path. But, honestly, how else do we learn? What other quality control checks do we have? If you find out that you were right- score! If you find out you were wrong, won't that help you with the next patient? You'll know how to be better next time. You'll think, I had another case like this and it turned out to be "x."

All of us strive to do our best for the lives that are put in our hands every day. Let us help you do just that.

- Heather Wyss, BVSc
(heather.wyss@ncagr.gov)

How to submit a Patient for a Necropsy:

- **Call us and let us know you're coming. We'll collect your information and request medical records.**
- **Anyone can submit an animal for necropsy— owners, staff, etc. There are also local couriers that will transport the patient to us.**
- **Whole animals should be cooled (not frozen) promptly after death and transported to us on ice.**
- **After the necropsy, remains can be picked up by a private crematorium or we can dispose of the remains at our lab.**

Salt (Sodium) Toxicity

By Dr. David Ackerman

Clinical History

On the afternoon, of August 19th Northwestern Lab received a phone call from an individual about a seizing boar hog. The owner had been checking his hogs that afternoon and found his boar unresponsive, acting blind, spinning in circles, foaming from the mouth and seizing. To see video, go to: <http://www.ncagr.gov/vet/ncvdl/newslettervideos.html> The hog died within an hour of the owner noting the condition and was presented to the lab that afternoon for a diagnostic necropsy. The 18-month-old boar hog was the sire of a small hog operation. During the breeding period, the sows and boar are maintained a pasture fed grass, a 16% feed ration, free choice water and minerals. Prior to presentation, the boar was removed from a group of sows a week earlier, dewormed with 1 % ivermectin and placed in an elevated, slatted floor house, where he normally resides except during the breeding period. Given this history and presentation the differentials included dehydration/salt toxicity, Rabies, Pseudorabies, aberrant parasite migration, trauma and other toxicities.

Gross Necropsy and Ancillary Tests

The 18-month-old boar hog weighted 240# (109kg). The gross necropsy revealed bilateral mild acute cranio-ventral congestion and firmness of the lungs and congestion of the meninges. The histopathology of the cerebrum documented encephalitis, mild to moderate multifocal eosinophilic perivascular cuffing. Other testing including Rabies results were negative. The diagnosis was salt toxicity.



Figure 1

Etiology of Occurrence

Salt (sodium) toxicity is the result of increased sodium ion concentrations in the blood stream of any animal; occurring in beef and dairy cattle, small ruminants, horses, dogs, cats, camelids, poultry and swine. Swine being the most susceptible. This can occur if an animal ingests a large amount of salt. However, more often salt toxicity occurs because of water deprivation or decreased water consumption. Animals deprived of salt or not accustomed to being fed salt, as part of their diet can overconsume feed or salt supplements if they are not acclimated to the change in diet. As mentioned, water deprivation or decreased water consumption is the most common reason for salt toxicity. Common events or factors that can lead to decreased water consumption include: overcrowding, isolation of an animal, placement in a new environment, unpalatable water, mechanical failure of waters, accidental failure to provide water, and frozen water sources. In practice, my experience was the last three events are the most common. Most of these circumstances can be prevented if appropriate management strategies are enlisted. With winter approaching, this is a great time for veterinarians to help clients evaluate water sources and management practices involving water availability.

Clinical Signs

Clinical signs can vary between species. However, there are some common symptoms among all the species which can be affected with this condition. The symptoms include; gastrointestinal signs such as diarrhea, excessive thirst, colic and in animals able, possible vomiting. Neurological signs are also common among species. They include, tremors, blindness, circling, weakness, and sudden

death. Other symptoms are typically species specific.

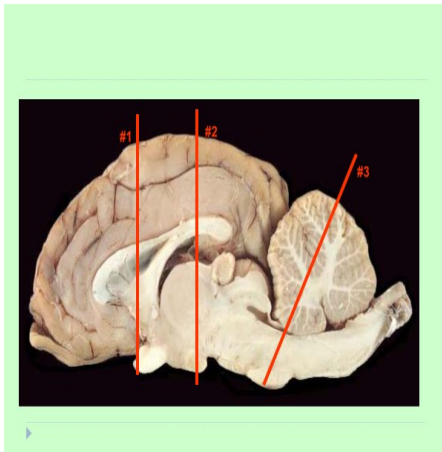


Fig. 2 Courtesy of Steve Rushton, DVM

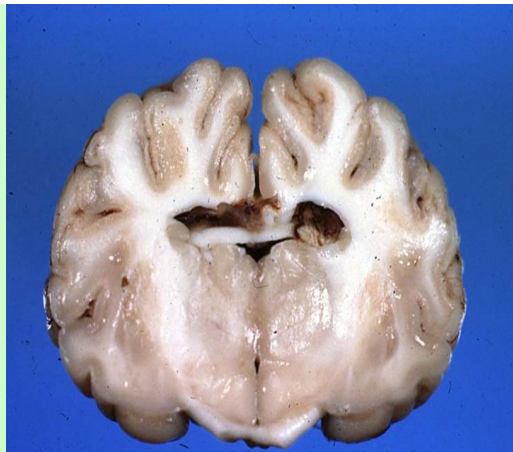


Fig. 3

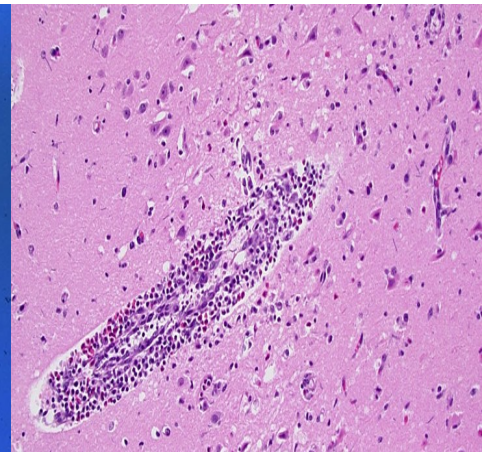


Fig. 4

Diagnosis

Unless the presumptive diagnosis is made based on history, and treatment immediately initiated, the diagnosis is often made by post mortem exam and diagnostic testing. For this discussion, we will focus on tissue sampling and testing in the post mortem case. In the case discussed, sampling for toxicology, bacteriology and histopathology are important. For toxicology retaining 5 g of the following organs; liver, kidney, brain and 100 g of stomach or rumen contents are necessary. In some circumstances, CSF or serum can be used for testing antemortem and post mortem. Check with the diagnostic lab you use for specifics, when determining the samples required for the tests you wish to have performed.

In the post mortem scenario, many times submission of brain tissue is advised as to the list differentials and the lesions which may or may not be found in the brain. Often, when submitting brain tissue, if not submitting the entire brain, a minimum of three sections of the brain from the areas diagrammed in Fig. 2 are necessary. One section from each area is required for histopathology. Section #1 gives a view of the cerebral cortex and internal capsule. Section #2 gives a view of cerebral cortex, hypothalamus and midbrain. While section #3 give a cross section of the cerebellum and brainstem. Two sections of cerebellum and brainstem would be required from area #3 if also performing rabies testing. Consult the pathologist at your diagnostic laboratory to determine how many additional sections and locations of brain may be required when working up a neurologic case. In the case of salt toxicity, general histological lesions often noted are inflammation, ulceration and congestion of the gastrointestinal tract, hydropericardium (in some species), edema of the cerebral cortex and laminar cortical necrosis (Fig 3). In swine, the laminar cortical necrosis occurs with eosinophilic perivascular cuffing (Fig 4), resulting in eosinophilic meningoencephalitis. In pigs, these lesions in the brain, typically develop in the first 48 hours of salt toxicity. However, in swine the eosinophilic perivascular cuffing has been documented to disappear in 72-96 hours. If a histopathological diagnosis cannot be determined, this may be where your samples for toxicology may be useful. With the list of symptoms varying between species for salt toxicity and numerous etiologies, compounded by the list of differentials with similar presentations, a good history, through examination and complete and correct acquisition of samples for testing are important for diagnosing the cause of disease.

Equine Lymphosarcoma

By Dr. Kimberly Hagans



A 16 year old, fully vaccinated, Warmblood gelding was presented to his veterinarian with a history of lethargy, inappetence, sheath and leg swellings, and low grade fevers. He was treated with a ten day course of antibiotics and Banamine. He responded but then relapsed when the therapy was discontinued. He became increasingly anemic and had petechiae. His RBC count decreased from 4.3 to 2.7 and his WBC count increased to 88,000. He was humanely euthanized.

The necropsy examination revealed pale and mildly icteric mucus membranes. Mild hepatomegaly with a nutmeg pattern in the hepatic parenchyma was present. The mucosal surface of the cecum and colon contained pinpoint raised white foci. The submucosa was thickened in the ileum and mild hyperemia was evident in the mucosa. The caudal abdominal lymph nodes were white-tan and nodular. The spleen completely filled the left side of the abdomen and wrapped around the stomach with the tail of the spleen filling the pelvic inlet. On the cut surface of the spleen, dry bulging splenic pulp was found. The spleen weighed 43 kg.

Histopathologic examination identified lymphosarcoma in the spleen and liver. Lymphoid leukemia was found in the bone marrow.

A normal horse spleen weighs between 15 and 20 pounds. This horse's spleen weighed 95 pounds. Equine lymphoma, also known as lymphosarcoma, is a form of cancer which involves the horse's lymphatic system. It most often starts in one lymph node but will eventually spread throughout the body.

There are many types of lymphoma. Typically, tumors are multi-centric and involve lymph nodes and thoracic and abdominal organs. They are most common in horses between 5 and 10 years old but can occur at any age. Equine lymphoma presents in four different anatomic forms: Generalized, Cutaneous, Alimentary, and Mediastinal.

Mesenteric Torsion

By Dr. Heather Wyss



A 5 year old, male, neutered, German shepherd service dog presented to an emergency clinic for acute vomiting and severe weakness. The dog had vomited four times through the night and was very weak in the morning. He arrested upon arrival to the ER. CPR was attempted but was unsuccessful.

The necropsy examination revealed black dilated small and large intestine. The lumen of the intestines were filled with black liquid. The root of the mesentery was twisted. Blood was coming out of the rectum. The perineal area was stained with blood.

A mesenteric torsion occurs when the intestines twist around the root of the mesentery. The cranial mesenteric artery becomes obstructed leading to ischemic necrosis. As the intestinal mucosa is damaged, blood flows into the lumen of the intestines giving bacteria and endotoxins entrance into the abdominal cavity and systemic circulation leading to circulatory and cardiogenic shock.

Clinical signs of a mesenteric torsion include weakness, vomiting, abdominal pain, recumbency, bloody stool, and the rapid onset of shock. Abdominal radiographs will show small intestinal distention and loss of abdominal detail.

Mortality rates are close to 100% even when caught early enough to surgical intervention. Many affected dogs expire during stabilization attempts before being taken to surgery.

The underlying cause for a mesenteric torsion remains unknown. German shepherds and German shorthair pointers are predisposed to this condition. Abnormal gastrointestinal motility secondary to exocrine pancreatic insufficiency, inflammatory bowel disease, intussusceptions, gastric dilatation volvulus, and gastrointestinal foreign bodies may be risk factors in the development of a mesenteric torsion.

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